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Detrended fluctuation analysis of heart intrabeat dynamics

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Abstract

We investigate scaling properties of electrocardiogram (ECG) recordings of healthy subjects and heart failure patients based on detrended fluctuation analysis (DFA). While the vast majority of scaling analysis has focused on the characterization of the long-range correlations of interbeat (i.e., beat-to-beat) dynamics, in this work we consider instead the characterization of *intrabeat* dynamics. That is, here we use DFA to study correlations for time scales smaller than one heart beat period (about 0.75 s). Our results show that intrabeat dynamics of healthy subject are less correlated than for heart failure dynamics. As in the case of interbeat dynamics, the DFA scaling exponents can be used to discriminate healthy and pathological data. It is shown that 0.5 h recordings suffices to characterize the ECG correlation properties.

Keywords: DFA; Intrabeat dynamics; Correlations

1. Introduction

The electrocardiogram (ECG) is a useful tool in cardiovascular system diagnosis. In medical practice, the specific waveform of heart beats recording at standard leads is of primary interest, because it is related to the electrophysiological processes are taking place to conduct impulses from the sinus node over the atria to the ventricles. An inspection of abnormalities or deviations in the ECG wave form helps to identify a large number of pathological conditions. Supplementary automatic methods have also been proposed by the scientific community, relying on a statistical analysis of a large number of heart beats intervals. Thus, the heart rate variability (HRV) is one of the most studied signals that provides certain statistical measures for detecting cardiac abnormalities [1]. Presumably, a strong relationship between the neuro-control autonomic systems and cardiovascular mortality, including sudden cardiac death, can be detected by the analysis of long HRV recordings [2]. Existing experimental evidence that show the propensity for lethal arrhythmia and signs of neuro-control malfunctioning has motivated studies for the development of autonomic activity measures. In this way, it is now accepted that HRV plays an important role for evaluating neuro-autonomic control and other influences. However, the human cardiovascular system is characterized by a high degree of complex variability, such that many standard measures obtained from HRV can also lead to incorrect conclusions and dangerous

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extrapolations [1]. As a consequence, in recent two decades we have witnessed the development of systematic procedures to characterize, both qualitatively and quantitatively, the HRV of healthy and unhealthy subjects.

Classical statistical analysis in time and frequency domains have widely explored, including the computation of means, standard deviations, histograms, power spectra distribution, etc. [3–5]. Of particular interest has been to use spectral analysis that shows dominant activities at low (around 0.1 Hz) and high (around 0.25 Hz) frequencies. In turn, these dominant dynamics have been related to the activity of the neuro-autonomic control, e.g., sympathetic and parasympathetic mechanisms [1,2]. Classical statistical analysis is based on the assumptions of linearity, stationary and equilibrium nature of HRV signals. Yet it has been proposed that methods borrowed from nonlinear analysis might also provide important insights for a physiological interpretation of HRV, and for the risk assessment of sudden cardiac death. In accord to actual nonlinear science trends, nonlinear dynamics and fluctuation analysis include the computation of Lyapunov exponents, Poincare sections, and fractal Hurst exponent [6]. Recently, detrended fluctuation analysis (DFA) has played an important role in the characterization of HRV dynamics [7], showing significant differences in the long-range scaling between heart beat dynamics in healthy and unhealthy subjects, [8] and between data from sleep and wake periods [6]. Multifractality features of HRV time series have been also explored, demonstrating the high complexity of heart rate signals [9]. Ashkenazy et al. [10] has used DFA to study magnitude and sign correlations in HRV as well, finding that magnitude time series are related to the nonlinear properties of the original beat-to-beat time series, while the sign series relates to linear properties. Kantelhardt et al. [11] have shown that interbeat ECG dynamics have very different DFA scaling characteristics during different sleep stages, which usually last from few minutes to half an hour. Karasik et al. [12] have shown significant scaling differences interbeat heart rate dynamics during rest and exercise epochs lasting between 6 and 10 min.

As a matter of fact, scaling analysis of HRV provides information, in the form of scaling exponents, of long-term correlations for time scales larger than the *mean* heart beat period. As mentioned above, it is presumed that such correlations reflect the feedback effect of neuro-controllers. However, a clinical analysis normally relies on voltage deflections within two heart beats since they are related to the heart's electro-physiological processes. Similarly to the analysis of HRV data, one can suspect the existence of a correlation pattern in the *intrabeat* (i.e., within one heart beat) dynamics, which can be used to characterize healthy and pathological data. In principle, such correlation pattern should become a signature of the cardiac electrophysiological processes. This paper uses DFA to study intrabeat correlations for time scales smaller than one beat period (about 0.75 s). Our results show that healthy intrabeat subjects dynamics is less correlated than the one of heart failure subjects. As in the case of interbeat dynamics [11,12], it is shown that (i) the DFA intrabeat scaling exponents can be used to discriminate healthy and pathological data, and (ii) that 0.5 h recording suffices to characterize the ECG correlation properties.

2. ECG data

We gathered 33 ECG recordings of the Physionet database (see the public internet site Physionet.org), which were collected from 18 healthy subjects and 15 patients with cardiac disfunction and were originally sampled with rates 128 and 250 Hz, respectively, at the Beth Israel Hospital. We used the congestive heart failure (CHF) database (chfdb) and the normal sinus rhythm database (nsrdb). The chfdb contains in total 15 patients with cardiac disfunctions, including three patients with atrial fibrillation (AF) cases (chf2, chf4 and chf6). The remaining 12 CHF patients correspond to 9 men, aged 22–71, and 3 women, aged 54–63. The nsrdb contains 18 healthy subjects (5 men, aged 26–45, and 13 women, aged 20–50).

3. Detrended fluctuation analysis

DFA is a widely used method to study long-term correlations in time sequences. For completeness in presentation, we provide a brief description of this method. For a given time series $y(t_i)$, $t_i = i\Delta t$, $i = 1, \ldots, N$, with sampling period Δt, the DFA method involves the following steps [7]:

1. Compute the time series mean $\overline{y} = (1/N)\sum_{j=1}^{N} y(t_j)$. An integrated time series $x(i)$, $i = 1, \ldots, N$, is then obtained as follows: $x(t_i) = \sum_{j=1}^{i} [y(t_j) - \overline{y}]$, $i = 1, \ldots, N$.

2. Divide the integrated time series $x(t_i)$ into boxes of equal size n, which corresponds to a time scale $\tau = n\Delta t$. A polynomial function of degree m, denoted by $x_{pol,m}(t_i;\tau)$, is used to interpolate the sequence in each box. The interpolating curve $x_{pol,m}(t_i;\tau)$ represents the local trend in each box.
3. Compute the fluctuation sequence as $z_m(t_i;\tau) = x(t_i) - x_{pol,m}(t_i;\tau)$, $i = 1,\ldots,N$. A linear fit (i.e., $m = 1$) is normally used.
4. The fluctuation function $F_m(\tau)$ is computed as the root-mean squared value of the sequence $z_m(t_i;\tau)$: $F_m(\tau) = \sqrt{(1/N)\sum_{j=1}^{N} z_m(t_j;\tau)^2}$.
5. Repeat the above procedure for a broad range of segment lengths n. According to the recommendations made by Peng et al. [7], the following range $n_{\min} \simeq 5$ and $n_{\max} \simeq N/4$ should be selected.

When the signal follows a scaling law, a power-law behavior for the fluctuation function $F(\tau)$ is observed:

$$F_m(\tau) \sim \tau^{\alpha_m},$$

where α_m is called the scaling exponent, a self-affinity parameter representing the long-range power-law correlation properties of the signal. In this way, the scaling exponent α_m is computed as the slope of the plot $\mathscr{F} = \{\log(\tau) \text{ versus } \log(F_m(\tau))\}$. In the case of having only short-range correlations (or not correlations at all) the detrended walk profile displays properties of a standard random walk (e.g., white noise) with $\alpha_m = 0.5$. On the other hand, if $\alpha_m < 0.5$ the correlations in the signal are *anti-persistent* (i.e., an increment is very likely to be followed by a decrement, and vice versa), and if $\alpha_m > 0.5$ the correlations in the signal are *persistent* (i.e., an increment is very likely to be followed by an increment, and vice versa). The values $\alpha_m = 1.0$ and $\alpha_m = 1.5$ correspond to $1/f$-noise and Brownian motion, respectively. A value $\alpha_m > 1.5$ corresponds to long-range correlations that are not necessarily related to stochastic processes. Indeed, $\alpha_m > 1.5$ can be reflecting deterministic correlations.

4. Results

Fig. 1 presents typical ECG waveforms from a healthy subject and a CHF subject. It can be observed that the waveform is qualitatively replicated on each beat, although some beat-to-beat variability can be observed. While reported scaling analysis of heart dynamics focuses on the variability of the beat-to-beat interval, this work uses DFA to study scaling for *intrabeat* dynamics (i.e., dynamics within heart beats). Since the beat-to-beat interval variance of all the cases studied is smaller than 20%, the maximum time scale to be considered was taken as 0.75 times the mean beat-to-beat interval of all ECG recordings. That is, the mean beat-to-beat value was computed for each record, and the maximum time scale was chosen accordingly. In this way, it can be guaranteed that the DFA scaling exponents extracted from the time sequence correspond to intrabeat correlations.

The DFA results (i.e., the data $\mathscr{F} = \{\log(\tau) \text{ versus } \log(F_m(\tau))\}$) display two different behaviors corresponding, respectively, to the cases 16 272 and 16 273 from Physionet database: (i) one that can be described with a unique scaling exponent (Fig. 2a), and (ii) another with a crossover accepting two scaling exponents $\alpha_1 < \alpha_2$, where α_1 is for smaller time scales and α_2 is for larger times scales (Fig. 2b). This observation motivated us to extract two parameters from each data set by fitting the scaling exponent over two different time scales. In this form, for each case the correlation results will be described by three parameters $\{\tau_c, \alpha_1, \alpha_2\}$ where τ_c is the time scale where a crossover occurring in the scaling exponent. Whenever the DFA data $\mathscr{F}$ can be described by a unique scaling exponent, we shall take $\alpha_1 = \alpha_2$.

Fig. 3 shows a plot of the scaling exponents α_1 and α_2, which were obtained by means of a least square fitting of the DFA data. The following can be observed:

1. Except four healthy cases, all CHF and AF cases presented a crossover. The crossover is very similar among different cases and its mean is about 0.21 s with variance 0.014 (see Fig. 4). Although the number of analyzed cases is limited by the availability of ECGs cases in Physionet database, this result suggests that the crossover could be an intrinsic property of many ECGs, regardless of the cardiovascular condition of the subject. It is apparent that the origin of this crossover and its position at this scale can be explained with

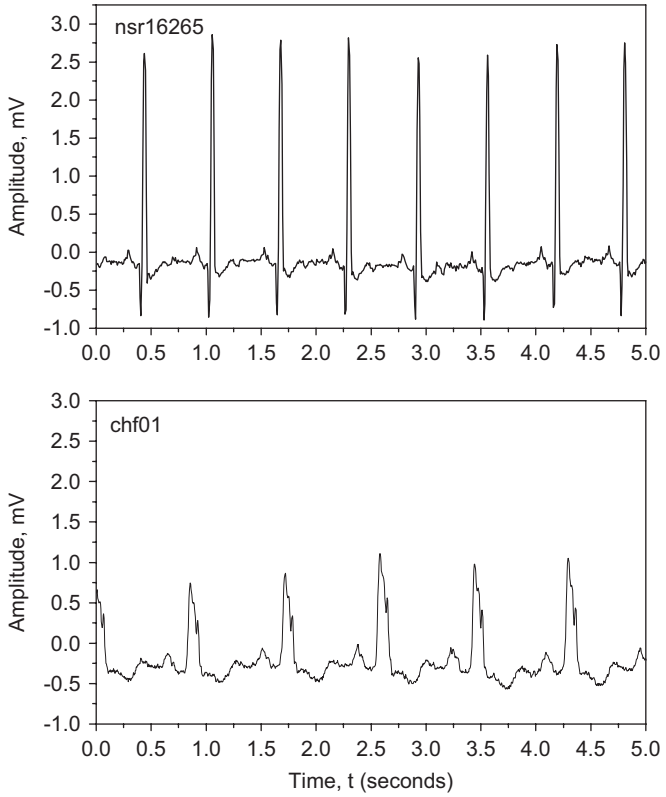

Fig. 1. ECG waveforms of healthy subject and a congestive heart failure patient.

the presence of repetitive periodic patterns formed by the P, Q, S and T waves in the intrabeat interval between two consecutive R peaks [13].

2. Despite that, only three AF subjects were studied, the results show no clear distinction between their intrabeat ECG dynamics and that for AF subjects. In fact, the scaling exponents α_1 and α_2 for AF subjects cannot be easily distinguished from those of CHF subjects.
3. In general, the scaling exponent α_1 is smaller for healthy subjects than for unhealthy CHF and AF subjects. In fact, one has that $\overline{\alpha}_{1,H} = 0.91$ with variance $\sigma_{1,H} = 0.14$ for healthy subjects, and $\overline{\alpha}_{1,U} = 1.46$ with variance $\sigma_{1,U} = 0.38$ for unhealthy subjects (both significantly with $p < 0.0001$). Notice that $\overline{\alpha}_{1,H} + \sigma_{1,H} = 1.05$ and $\overline{\alpha}_{1,U} - \sigma_{1,U} = 1.08$. That is, $\overline{\alpha}_{1,H} + \sigma_{1,H} < \overline{\alpha}_{1,U} - \sigma_{1,U}$ such that, up to the variances $\sigma_{1,H}$ and $\sigma_{1,U}$, the short-range scaling exponent α_1 is able to discriminate between healthy and unhealthy conditions.
4. While the low-range scaling exponent α_1 seems to be a good parameter to discriminate data between healthy and unhealthy subjects, the large-range scaling exponent α_2 displays no significant differences between such conditions. In fact, one has that $\overline{\alpha}_{2,H} = 0.59$ with variance $\sigma_{2,H} = 0.17$ for healthy subjects, and $\overline{\alpha}_{2,U} = 0.89$ with variance $\sigma_{2,U} = 0.22$ for unhealthy subjects. Here, $\overline{\alpha}_{2,H} + \sigma_{2,H} = 0.75$ and $\overline{\alpha}_{2,U} - \sigma_{2,U} = 0.67$, so that $\overline{\alpha}_{2,H} + \sigma_{2,H} > \overline{\alpha}_{2,U} - \sigma_{2,U}$. In terms of relative differences with respect to healthy cases, one has that

$$\frac{(\overline{\alpha}_{2,H} + \sigma_{2,H}) - (\overline{\alpha}_{2,U} + \sigma_{2,U})}{(\overline{\alpha}_{2,H} + \sigma_{2,H})} \times 100 = 11.41\%.$$

That is, deviations larger than about 11.41% relative to the mean for healthy cases could be considered as candidates for unhealthy condition. As before, given the limited number of studied cases, this result suggests that the long-range scaling exponent α_2 is not as good as α_1 to discriminate between healthy and unhealthy conditions.

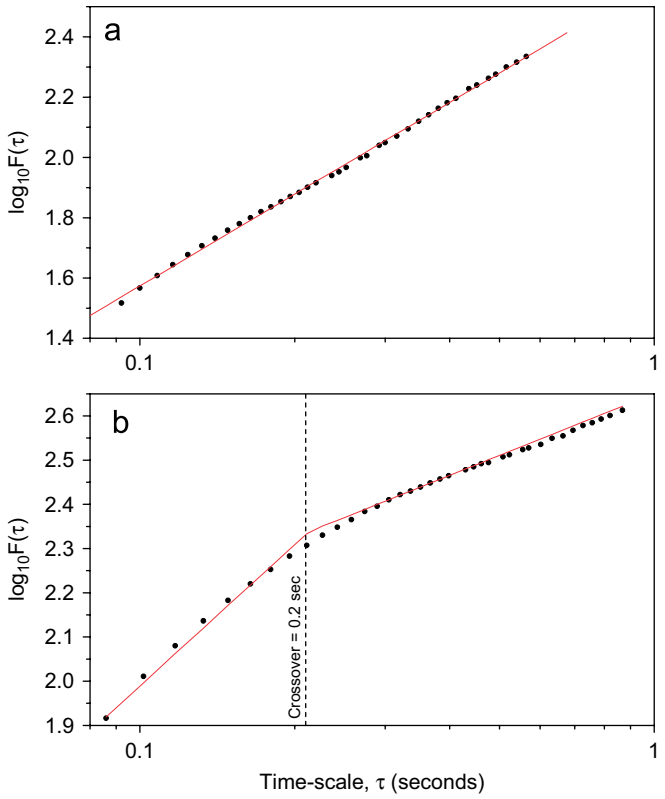

Fig. 2. DFA results show two typical behaviors: (a) one that can be described by a unique scaling exponent; and (b) another with a concave crossover.

As noted above, healthy subjects display less correlated intrabeat dynamics than CHF and AF subjects. This finding is consistent with previous interbeat analysis reports of altered correlation properties under pathological conditions [7].

4.1. Discussion

Regarding the results described above, the following comments are in order:

1. It can be appreciated that the data from healthy subjects are tightly clustered suggesting that there may exist a universal scaling behavior for intrabeat dynamics. In contrast, the pathological data show more variation, which may be related to different clinical conditions and vary according to the severity of the pathological states.
2. HRV depends strongly on the age of the subjects; younger individuals have a significantly higher HRV than elderly ones [14]. Although it is clear that age has a negative effect on the variability response of the cardiovascular system, this dependence may be attributable to different levels of daily activity: in general, younger subjects have a larger variety of physical activity than elderly ones, which is supported by cardiovascular system with a relatively wide operation range. Since a scaling analysis of intrabeat dynamics is normally based on long-time recordings of approximately 8 h, it is very likely that sections with different mean beat-to-beat value be contained in the HRV recordings. The results presented in this paper are based on 30 min ECG time series, for which we checked that no major variations of the mean heart rhythm were manifested. The effect of biases induced by daily activity variations are reduced and, in principle, the scaling exponents α_1 and α_2 reflect some features of the intrabeat correlations of the cardiovascular system.

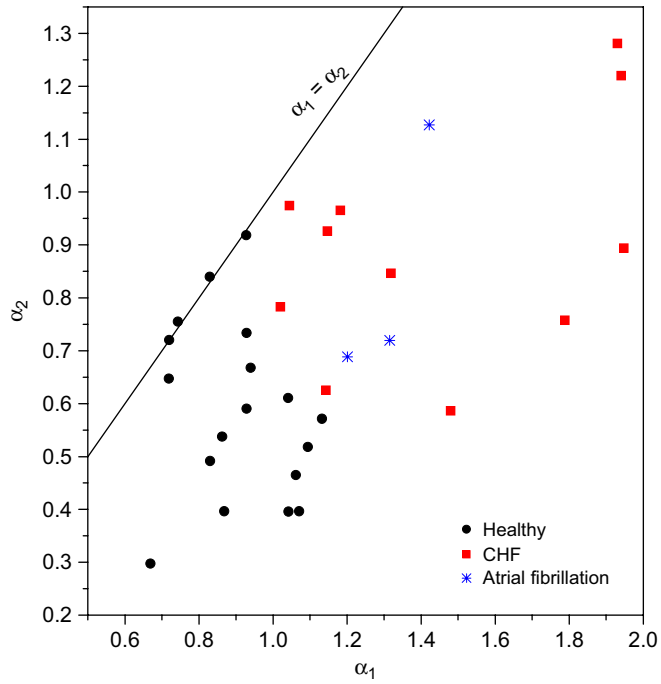

Fig. 3. Scatter plot of scaling exponents α_1 and α_2 for healthy and unhealthy subjects. The α's were calculated from intrabeat dynamics of 30 min ECG recordings. Notice good separation between healthy and heart disease subjects, with clustering of points in two distinct clouds.

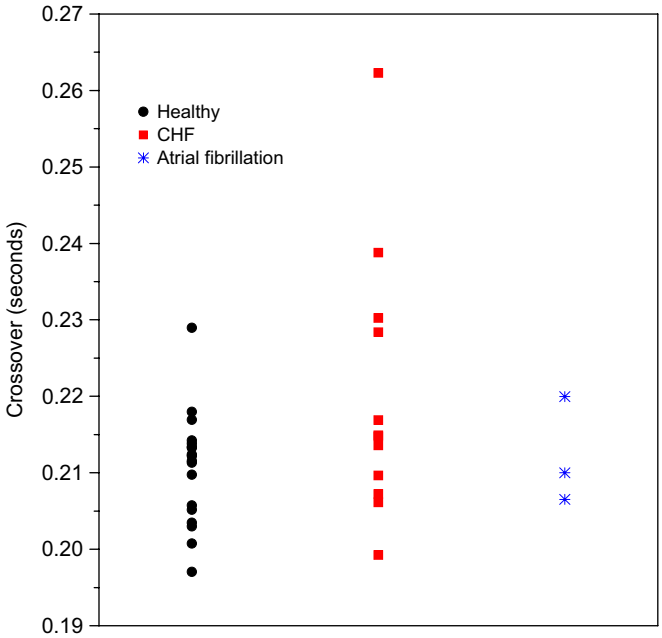

Fig. 4. Crossover for healthy and heart disease conditions. Notice that the low variability of the crossover in all cases, which may be suggesting that the crossover is an intrinsic property of intrabeat ECG dynamics.

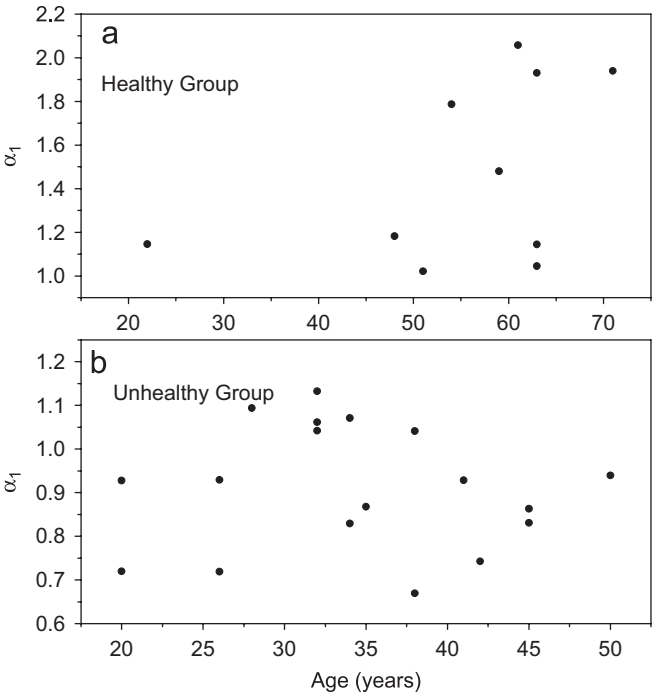

Fig. 5. Short-range scaling exponent α_1 relative to the age of the subjects; similar results are found for α_2. Notice the lack of correlations between age and scaling exponents for both (a) healthy and (b) unhealthy conditions.

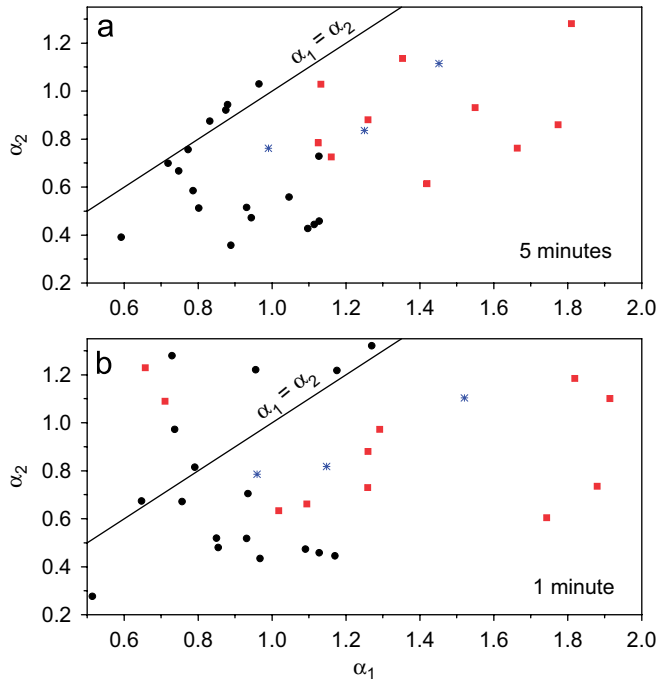

Fig. 6. Scatter plot of scaling exponents α_1 and α_2 for healthy and unhealthy subjects. The α's were calculated from intrabeat dynamics of duration: (a) 5 min; and (b) 1 min. Notice that the 5 min case still retains the scaling and separation characteristics shown by the 30 min case.

Fig. 5 displays the short-range scaling exponent α_1 relative to the age of the subjects; similar results are found for α_2. For both healthy and unhealthy subjects, the lack of a significant effect resulting from age on the intrabeat scaling exponents is observed. This shows that the observed ECG scaling differences are produced by the cardiovascular condition rather than by the effect of subject age. That is, the effects of the cardiovascular unhealthy condition seems to be dominant over the adverse age effects in the intrabeat variability. This is an interesting result since, given the relatively short duration ECG time series requirement, intrabeat scaling exponents can recover the actual cardiovascular correlations (i.e., without the effects of exogenous disturbances and changes of physical activity), which by contrast can introduce the non-stationarities that are found in long-time HRV data (for instance, 24 h time series in Peng et al. [7] and Wessel et al. [15]).

3. It has been shown that 30 min ECG time series may be adequate to characterize, at least statistically, intrabeat dynamics of healthy and unhealthy conditions. For practical purposes, clinical investigators are usually interested in the possibility of using substantially shorter time series. Fig. 6 shows the scaling exponents α_1 and α_2, for 5 and 1 min time series. Observe that the 5 min case retains the results shown in Fig. 4 without serious deformation. However, 1 min time series are too short to retain the intrabeat variations and discriminate between healthy and unhealthy conditions. Based on these results, we arrived to the conclusion that time series of short duration (less than about 3 min) are unable to retain the variability of the ECG wave. Since ECG measurements are relatively inexpensive with current clinical equipment, it is economically reliable to characterize unhealthy cardiovascular states with time series with duration of at least 10 min. The scaling exponent indexes are persistent over long-time records. In fact, Figs. 7 and 8 show, respectively, the evolution of the short-term scaling exponent α_1 for a 5 and 30 min window with 80% overlapping, over a 18 h ECG record. Notice the clustering of healthy and unhealthy cases over the whole time period. From these results, it is apparent that increased scaling exponents for unhealthy subjects are persistent despite variations in the day and night activities.

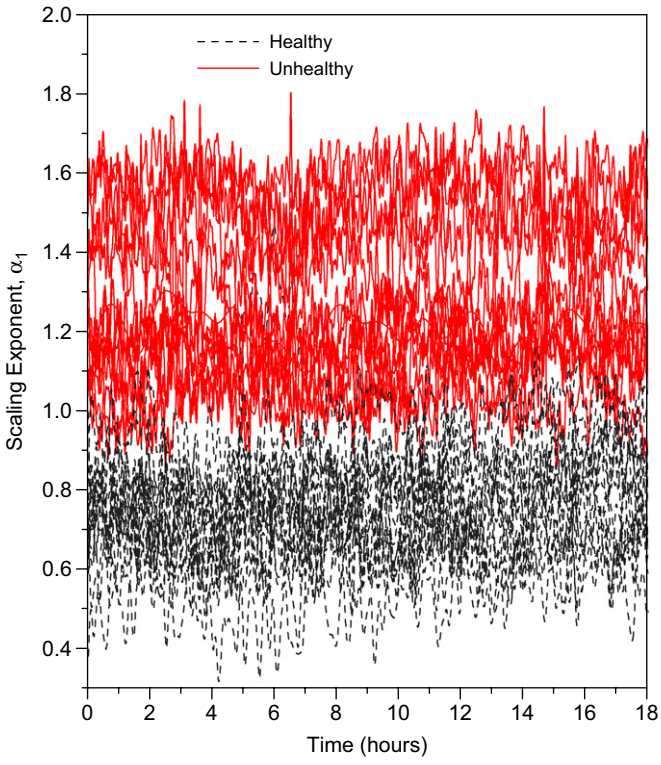

Fig. 7. Evolution of the short-term scaling exponent α_1 for a 5 min window, over a 18 h ECG record. Notice the clustering of healthy and unhealthy cases over the whole time period.

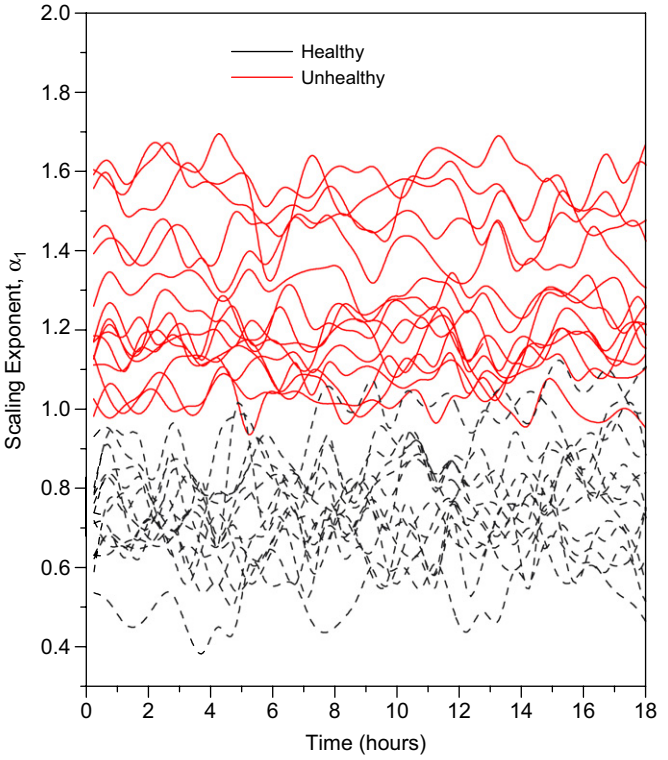

Fig. 8. Evolution of the short-term scaling exponent α_1 for a 30 min window, for the ECG records in Fig. 7. As above, the presence of a clustering of healthy and unhealthy conditions is apparent.

4. The origin of the increment of correlation under pathological conditions is unclear. However, it can be related to a loss of complexity of the dynamics of cellular activation and repolarization properties of the heart tissue. In fact, Kim et al. [16] have shown that a decrease in the number of wave fronts in ventricular fibrillation by tissue mass reduction can cause a transition from chaos to quasi-periodicity. The mechanisms by which the number of wave fronts decreased can be attributed to a reduced heart tissue mass, which in turn reduces the number of invading polarization wave fronts. When the boundary to mass ratio is increased by, e.g., CHF effects, it is more likely for the reentrant wave front to terminate by arriving at a boundary, which reduces the complexity of the polarization dynamics [16]. In terms of DFA, this is reflected as increased intrabeat scaling exponents α_1 and α_2.

5. Conclusions

In summary, we have applied DFA to ECG time series from healthy subjects and patients with heart diseases (congestive heart failure and atrial fibrillation). We show that the intrabeat dynamics displays differences in the scaling behavior of healthy and unhealthy subjects over time scales smaller than about 0.75 times the mean beat period. Contrary to scaling analysis of interbeat (i.e., HRV) dynamics that require very long-time series (at least 8 h), our results suggest that significant differences in scaling of intrabeat dynamics can be appreciated with time series of about 5–30 min, thereby making intrabeat scaling analysis potentially applicable to real clinical data.

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